IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF TEXAS
HOUSTON DIVISION

BEN N. BROWN, SR.	§	
	§	
Plaintiff,	§	
	§	
v.	§	
	§	C.A. NO. 4:08-cv-00413
SHELL OIL COMPANY, SHELL CHEMICAL,	§	
L.P, RADIATOR SPECIALTY COMPANY,	§	ADMIRALTY
AND JOHN DOES 1 THROUGH 250,	§	
INCLUSIVE,	§	
	§	
Defendants.	§	

PLAINTIFF'S MEMORANDUM IN SUPPORT OF OPPOSITIONS TO MOTIONS OF DEFENDANTS TO EXCLUDE PLAINTIFF'S EXPERTS AND FOR SUMMARY JUDGMENT

Plaintiff, Ben N. Brown, Sr., opposes the following defense motions on the grounds set forth in the subjoined Points and Authorities:

1. *Shell Defendants' Motion for Summary* Judgment (subject to Shell Defendant's Motion to Exclude Plaintiff's Causation Experts), joined by Defendant Radiator Specialty Company ("Radiator");

2. *Shell Defendant's Motion to Exclude Plaintiff's Causation Experts*, joined by Radiator Specialty Company.

3. *Shell Defendants' Motion To Exclude The Testimony Of Plaintiff's Expert Melvyn Kopstein*.

The motions of Shell and Radiator Specialty Company to exclude Plaintiff's special causation experts, Melvyn Kopstein and Mark Nicas, are opposed as premature to the disposition of the foregoing motions since the issues are distinct and not ripe for decision. The court has denied Radiator's motions, joined by Shell, but Shell's independent motion as to Melvyn Kopstein remains pending. Out of an abundance of causation, Plaintiff attaches Melvyn Kopstein's declaration supporting his methodology through Shell's own reports.

Points and Authorities

Defendants Shell Oil Company, Shell Chemical, L.P and Shell Offshore, L.P. ("Shell Defendants" or "Shell")[1] have moved to exclude the testimony of Plaintiff's experts who will testify that exposure to materials and products containing benzene causes multiple myeloma ("MM"), the disease Plaintiff contracted while employed as a seaman by Shell. Shell's Motion for Summary Judgment acknowledges that if its motion to exclude fails, its companion to, then the summary judgment motion of necessity fails as well. Plaintiff's proof of the specific causation, that his benzene exposure while employed by Shell caused, in whole or in part, his MM.[2]

Shell's predicate Motion to Exclude Plaintiff's Causation Experts attacks the admissibility of three of Plaintiff's retained experts: Peter Infante, Dr.PH, Sheila L. Butler, MD, MPH and Robert J. Harrison, MD, MPH. These experts base their opinions on their review of epidemiological studies, toxicological studies, evidence of biological plausibility for benzene to induce MM, other data, standard reference works, commentary by government agencies and other scientific bodies concerned with studying disease. The experts' opinions are based on sound methodologies and, as required by FED. R. EVID. 702 for admission, "based upon sufficient facts or data," and are the "product of reliable principles and methods."

Shell's motion reveals that it fails to appreciate, or even to state accurately, the facts of Mr. Brown's exposure to benzene while he worked for Shell. Also, the motion misstates the standard for judicial review of expert testimony about the causes of disease in a toxic tort suit. The fact that Plaintiff's experts' opinions differ with those of the Defendants' experts does not support a motion to exclude the testimony of the Plaintiff's experts on causation. Genuine

[1] Plaintiff added as a named defendant Shell Offshore, Inc., a wholly-owned subsidiary of Shell Oil Company, by Second Amended Complaint filed on February 24, 2009.
[2] See Shell's Motion for Summary Judgment, p. 1 and p. 10, para. 10.

material issues of fact exist as to benzene's causation of MM and as to the significant and harmful levels of exposure to Mr. Brown of materials containing the known carcinogen benzene.

Plaintiff's three general causation experts should be permitted to give testimony on the issue of general causation.[3] These experts have rendered preliminary opinions and further opinions in support of this response that are both reasonable and reliable based on sound accepted methods of analysis of epidemiological evidence indicating significantly more than the two-fold statistical evidence of increased risk for workers who had similar exposures to benzene containing materials in the same and similar occupations. Their opinions address the rate of error in analysis and explore possible other causes for the plaintiff's disease, all providing further evidence pointing to the plaintiff's Shell work experience as the most likely source of his disease.

I. Statement of Contested Issues of Material Fact

1. Plaintiff's expert opinions that benzene containing materials can cause the disease multiple myeloma are well founded and employ reasonable standard methodologies that are generally accepted.

2. The studies the Plaintiff's causation experts rely upon support the reasonable inference that benzene can cause MM. The general causation opinions are supported by reliable cohort and case control epidemiological studies involving benzene exposure and made employing accepted and standard methodologies. The Plaintiff's expert's opinions are further supported by a peer-reviewed[4] meta-analysis conducted by Dr. Infante. See Exhibit A, *Benzene Exposure and Multiple Myeloma: A Detailed Meta-analysis of Benzene Cohort Studies* (2006). This meta-analysis is a study collecting appropriate cohort studies of workers exposed to

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[3] These three experts also offer opinions that Mr. Brown's exposure to benzene by Shell specifically was a contributing cause of his MM.

[4] See Exhibit B.

benzene, which provided further evidence of the increased risks of such workers contracting MM.

3. The Plaintiff's expert's general causation opinions are supported in several studies of oil and gas production workers, who like Plaintiff, worked with, and had direct contact with, crude oil and other oil and gas produced materials containing significant amounts of benzene. Their opinions also include explanations of the biological plausibility of the association of benzene exposure and the development of MM.

4. Plaintiff was exposed to significant and harmful levels of benzene contained in the products and materials he used while working for Shell, and the studies support scientific inferences that benzene exposure caused or significantly contributed to his MM.

II. Standard for Review of Expert Opinions in Toxic Tort cases

In *Daubert v. Merrell Dow Pharmaceuticals*, 509 U.S. 579 (1993) the Supreme Court directed federal trial courts to set aside their reliance on prior opinions, primarily *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923), which promoted the standard of admissibility of expert opinions that they be "generally accepted," and instead to rely on the Federal Rules of Evidence to evaluate before trial the reliability and relevance of expert scientific opinions. As the Supreme Court pointed out, the "generally accepted" standard relied upon by Shell is not found in FED. R. EVID. 702.

A challenge to the admission of expert opinion testimony, according to *Daubert*, is to be guided by FED. R. EVID. 104 (a) and 702. As is generally understood, those rules require the trial judge to make a preliminary determination whether a "witness qualified as an expert…may testify" by determining if the testimony will "assist the trier of fact to understand the evidence or to determine a fact in issue" and "if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principals and methods, and (3) the witness has applied

the principals and methods reliably to the facts of the case."

The *Frye* court holding that expert opinion testimony must be "generally accepted" in order to be admitted into evidence was deemed by *Daubert* to be too restrictive, causing some judges to assume that an opinion that varied from the generally accepted view may not be reliable and therefore not admissible, and promoted the notion that only opinions of general consensus among experts in a given field were worthy of consideration by the triers of fact. Yet, Shell's contentions articulated in its Motion to Exclude Plaintiff's Causation Experts are that the opinions of Plaintiff's three general causation experts are not generally accepted and the patently false statement that "[t]here is no evidence of general causation between exposure to crude oil, condensate, solvents, or paints and multiple myeloma." *Daubert* specifically held that divergence of opinion did not disqualify an expert who has arrived at conclusions that might differ from the majority opinion on a subject. The reliability and soundness of the methods used by the expert in support of her opinion could be subject to challenges, not the degree to which that opinion departed from any consensus that may exist among those deemed to be expert in a field of scientific knowledge.

In order to evaluate proffered expert opinion and determine whether a jury hears the contested opinion testimony, *Daubert* recommended that the trial judge weigh the following factors:

1. The challenged expert opinion typically should be evaluated to learn if it can be tested. Can the hypothesis be proved false? Or, is the opinion one that can survive scrutiny even if it is not recognized as reflecting a consensus view of recognized experts? Epidemiological studies of persons similarly situated as Plaintiff with similar occupational exposure to toxins associated with disease should be evaluated through the expert's opinion. Animal studies may shed light on the plausibility of the expert's opinion about the cause of the disease in question.

And, other kinds of tests may be available to measure the strength of the associations between the disease causing agent and the disease may be available for the expert in reviewing inferences leading to evidence concerning the subject.

2. The trial judge can look for support for the challenged opinion in peer reviewed works and journals and published articles. These factors may support an opinion but, the absence of every support concerning these factors does not cause the opinion to be inadmissible.

3. The trial court should also attempt to learn if the expert has been able to assign a rate of error in the opinion he has reached which takes into account variables that may impact the opinion and which may or not affect the reliability and strength of the opinion. Logically and frankly addressing the rates of error should help to support an opinion. This assignment of a rate of error and a range of values may help to show further reliability for the opinion.

4. "Finally, 'general acceptance' "can yet have a bearing on the inquiry. A "reliability assessment does not require, although it does permit, explicit identification of a relevant scientific community and an express determination of a particular degree of acceptance within the community."

Under the *Daubert* framework then, a trial court should not require general acceptance of the causal link between an agent and a disease or condition if the expert otherwise bases his or her opinion on data and facts employing a reliable methodology.

Review of the opinions of Drs. Infante, Harrison and Butler employing these factors reveal that their opinions are well founded, reliable and pertinent to the jury's task of determining whether benzene can cause the MM Mr. Ben Brown suffers from, and if it likely did cause that disease.

A. Applicable *Daubert* Jurisprudence

Subsequent to *Daubert*, the Supreme Court admonished federal trial court judges that

their "role as evidentiary gatekeeper is not intended to replace the adversary system but to ensure that "an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practices of an expert in the relevant field." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 119 S. Ct. 1167, 14 L. Ed. 2d 258 (1999). The task then of the trial court requires a careful examination of the scientific methods used by the expert in reaching an opinion so that it is both reliable and relevant. *General Electric v. Joiner*, 522 U.S. 136 (1997). The "focus, of course, must be solely on principals and methodology, not on the conclusions they generate." *Daubert v. Merrell Dow Pharmaceuticals*, 509 U.S. at 595..

Rule 702 is broad enough to permit testimony that is the product of competing principals or methods in the same field of expertise. See *Heller v. Shaw Industries, Inc.*, 167 F.3d 146, 160 (3d Cir. 1999) (expert testimony cannot be excluded simply because the expert uses one test rather than another, when both tests are accepted in the field and reach reliable results). As stated in *In Re Paoli R.R. Yard PCB Litigation*, 35 F.3d 717, 744 (3d Cir. 1994), parties do not have to demonstrate to the trial judge by a preponderance of the evidence that the assessments of their retained experts are correct; they only have to demonstrate by a preponderance of the evidence that their opinions are reliable. "The evidentiary requirement of reliability is lower than the merits standard of correctness."

B. The Federal Judiciary Center "Reference Manual on Scientific Evidence"

Foremost among the references available to guide the trial judge in this work is the Federal Judiciary Center's sponsored guide to the review of scientific evidence published in 1998 and revised in a second edition in 2000. The Federal Judicial Center Reference Manual on Scientific Evidence was provided by the Judiciary Center to assist the federal courts in the review of the testimony proposed by parties seeking to have expert scientific testimony admitted

into evidence in the trial of toxic tort cases. The manual includes a "Reference Guide on Epidemiology" (attached as Ex. A hereto), another on the subject of Toxicology and a third on Medical Testimony. The recent decision by the Nebraska Supreme Court in *King v. Burlington Northern Santa Fe Ry. Co.*, 277 Neb. 203 (2009) makes extensive use of the "Reference Manual on Scientific Evidence, Guide on Epidemiology" (hereafter "Epidemiology Guide"), a decision that concerned a *Daubert* type challenge of the plaintiff's expert opinion on benzene induced multiple myeloma in a case based on the Federal Employee Liability Act, which is also the basis of the maritime personal injury Jones Act claims in Mr. Brown's case. The Nebraska court follows an adoption of the *Daubert* standard and their evidence code tracks the federal rules. *King v. Burlington Northern Santa Fe Ry. Co.*, 277 Neb. 203, 207, 229 (2009). The King opinion provides persuasive authority to for the use of the Epidemiology Guide in evaluating *Daubert* challenges and is attached as Ex. B to this memorandum.

Epidemiological studies play a key role in identifying evidence of general causation. The plaintiff must prove in a toxic tort case that a substance, in this case materials containing benzene, is capable of causing a particular injury, such as Mr. Brown's MM. In *Knight v. Kirby*, 482 F. 3d 347, citing *Merrell Dow Pharmaceuticals, Inc. v. Havner*, 953 S.W. 2d 706, 714 (Tex. 1997), the court stated "General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual's injury." The introduction to Epidemiology Guide provides, "Epidemiology is the field of public health and medicine that studies the incidence, distribution, and etiology of disease in human populations. The purpose of epidemiology is to better understand disease causation and to prevent disease in groups of individuals. Epidemiology assumes that disease is not distributed randomly in a group of individuals and those identifiable subgroups, including those exposed to certain agents, are at increased risk of

contracting particular diseases." Epidemiology Guide, p. 335.

Epidemiological studies can be useful in identifying an agent causing a disease and may reveal associations between a cause and an effect even when such studies do not definitively prove that relationship. *Ambrosisni v. Labarraque*, 101 F.3d 129 (D.C. Cir. 1996). These studies may provide the basis for an expert to draw reasonable inferences supporting his opinion about general causation and provide foundations for the expert's conclusions. The proper purpose of "[t] he epidemiological studies [is to] examine existing populations to attempt to determine if there is an association between a disease or condition and a factor suspected of causing that disease or condition" *Merrell Dow Pharmaceuticals v. Havner*, 953 S.W. 2d 706, 715 (Tex. 1997).

As can be seen from the attached affidavits of Plaintiff's three causation experts, their opinions are based on valid epidemiological studies. That Defendants can produce experts with countervailing opinions is not a basis for exclusion.

C. Epidemiology Guide Factors

The Epidemiology Guide provides a thorough list of nine factors, which include the *Daubert* factors. Epidemiology Guide at pp. 375-379.

1. There must be a *temporal relationship* between exposure to the suspected agent (i.e. carcinogen) and then later development of the disease. There is ample evidence that Mr. Brown was exposed to benzene containing materials starting in 1968 and continuing through 1990. This period of exposure, 39 years from first exposure to diagnosis and 17 years from last exposure to diagnosis, is consistent with the period of latency[5] of MM. See affidavits of Peter F. Infante ("Infante") ¶¶ 7, 75; Sheila L. Butler ("Butler") ¶ 20; Robert J. Harrison ("Harrison") ¶1.

2. The strength of association measured by a *relative risk* showing the increased risk

[5] The period between first exposure to the carcinogen and the first diagnosis of the disease.

of contracting the disease among the exposed and non-exposed individuals. The threshold for concluding that an agent more likely than not caused a disease is expressed in a relative risk ratio ("RR") of more than 2.0, meaning a twice greater risk of exposed persons contracting the disease than a the general population of unexposed persons. When the RR is expressed as more than 2.0, courts typically appreciate that this is evidence that the disease more likely than not was caused by demonstrated exposure to the agent in the absence of contrary evidence, such as an alternate source of exposure. The affidavits of Infante, ¶¶ 13, 19, 62, 64, 69 and 73; Butler ¶ 17, and Harrison ¶ 2 demonstrate RR well above 2.0, as high as 5.0, for MM in many of these studies.

3. A *dose-response relationship* between exposure and disease is a strong, but not essential factor, and often is the most elusive. Human experimentation studies using people exposed to benzene are unethical, and threshold minimum risk levels of exposure are not reliable. There may be no safe minimum exposure to benzene. The American Petroleum Institute reported in a 1948 that there may be no absolutely safe level of benzene exposure because of its capacity to suppress and damage blood cell formation in the bone marrow. Animal studies can be helpful but not conclusive in identifying dose-response. Affidavits of Infante ¶¶ 59, 62, 64, 69, 73; Butler ¶¶ 8, 9, and Harrison ¶ 3.

4. *Replication of findings* is demonstrated when similarly exposed populations reveal similar rates of disease. Such associations are described and explained in the affidavits of all three of Plaintiff's retained general causation experts, *in extenso*. See, e.g., Harrison ¶ 4.

5. *Biological plausibility factors* establish the consistency between current biological knowledge and the biomarkers and other evidence that benzene has a disruptive and harmful impact upon blood cell formation and disease processes. Such associations are described and explained in the affidavits of Infante ¶¶ 29 – 44, and Harrison ¶ 5.

6. The consideration of *alternative explanations* for the occurrence of the disease is

important to identify and to rule out bias, other confounding circumstances ("confounders"), data errors and other error affecting the analysis and methods used to inquire about rates of disease and exposure to agents. Such associations are described and explained in the affidavits of Butler ¶¶ 10 – 13, 22, 24-29, and Harrison ¶ 6..

7. The *cessation of exposure* invokes the question whether a halt in further exposure may result in reduced rates of disease. Such data is often unavailable. Such associations are described and explained in the affidavit of Infante ¶ 19; Harrison ¶ 7.

8. *Specificity of exposure* may be evident when an agent causes a single disease or similar set of closely related diseases. Benzene exposure is regarded as a carcinogen impacting the blood cell forming tissue and adversely affecting many blood cell types. Benzene is implicated in a number of cancers that involve disruption of blood forming tissues in the body. Such associations are described and explained in the affidavits of Infante ¶¶ 29, 43; Harrison ¶ 8.

9. *Consistency with other knowledge* inquires whether there is information which may explain rates of disease and other aspects of exposure to physical agents and disease. Benzene production increased dramatically and exponentially in the first decades of the 20th century, and disease associated with disruption of blood formation appeared for the first time in significant amounts thereafter. In particular the increase in MM after 1960 is associated with more widespread exposure to benzene containing materials in the workforces and the general population. Such associations are described and explained in the affidavit of Harrison ¶ 9.

D. Shell Assertions Contested

1. Plaintiff contests Shell's assertion that Mr. Brown was exposed to only small amounts of benzene.

Shell is wrong in asserting that Mr. Brown was exposed to "small amounts of benzene" (Shell Brief on Causation Experts, p.1) The type of work the plaintiff did, mostly taking place out-of-doors with some work doing tank cleaning and gauging in enclosed spaces, is similar to

the work described in the epidemiological study done of Norwegian offshore oil and gas production workers, and in other studies of oil and gas production workers cited below and discussed by Dr. Infante in his affidavit. Furthermore, these studies reveal that workers' exposure to materials containing relatively small amounts of benzene experienced significantly increased risks of contracting multiple myeloma compared to the general population not having such work place exposures. Ben Brown spent his work life with materials containing significant amounts of benzene. His exposure is similar and most likely greater than that described in the epidemiological study concerning offshore oil workers exposure to crude oil, which found evidence of increased risks of MM and acute myelogenous leukemia ("AML"), studies of merchant seamen exposed to crude oil on oil tank ships and other crude oil studies, each involving exposure to benzene as a naturally occurring constituent of crude oil.[6] Mr. Brown was a maintenance worker on boats, barges, and oil well platforms. As such, he had direct contact with crude oil with benzene content - according to Shell records, ranging from 0.15% to 0.3%, or 1,500 ppm to 3,000 ppm benzene. Crude oil contact was frequent and involved many tasks including oil spill clean-up, burning waste crude oil, opening pipes and valves, contact with mixtures of oil, and contact with paraffin waxy deposits inside well pipe, usually mixed with organic solvents used to cut out the paraffin and other waxy build up inside well pipe.

Shell's oil production manuals called for using solvents such as reformate to do this work. Shell Oil material data safety sheets describe reformates with benzene content ranging from 1% to more than 10% benzene (10,000 ppm to 100,000 ppm benzene). According to Shell

[6] "Increased Risk of Acute Myelogenous Leukemia and Multiple Myeloma in a Historical Cohort of Upstream Workers Exposed to Crude Oil", Kirkeliet, J., Riise,T., Bratveit, M. and Moen, B., *Cancer causes control* (2008) 10:13-23; "Benzene Exposure and Hematological Effects Among Offshore Workers Exposed to Crude Oil," Jorunn Kirkeleit, *Dissertation for the degree philosophiae* (PhD) at the University of Bergen (2007); crude oil studies cited by Dr. Infante, Divine, BJ and Hartman, CM, "Update of Crude Oil Production Workers 1946-94", Occup. Env. Med. 57, 411-417; and, a case control unpublished study of Union Oil workers concerning leukemia, non Hodgkin's lymphoma and multiple myeloma submitted to Union Oil Co. in 1992. Plaintiff's expert Mel Kopstein, a PhD and chemical engineer, notes studies describing levels of benzene exposure on oil tankers carrying crude oil cargoes

records the commercially available solvent made by Shell most like "reformates," with similar physical characteristics such as boiling point ranges and rates of evaporation, was Shell Sol B, which contained 0.74 % benzene (7,500 ppm benzene). The solvents were used by Mr. Brown to do all manner of cleaning of oil-fouled decks at platforms, cleaning tools, doing the paraffin cutting work, any other job when oil had to be cleaned up, and, especially troubling, it was a practice of these workers and Mr. Brown to use the solvents to clean oil and tar off their hands and arms. Shell has admitted that it produced all of these or the vast majority of these solvents and provided them to its offshore oil and gas production workers. Use of solvents for the paraffin cutting work was done by Mr. Brown on a daily basis for a year to two years between 1969 and 1974. Brown's testimony, pp. 53, lines 1-8; 54, lines 19-23. This work consumed his time daily, *Id*., 61, line 22, and it took all of his 12 hour duty time, but for travel to and from the wells he worked. *Id*. 64, lines 3-6. He testified he would smell it, his gloves were wet with the solvent, and he got it on his body. *Id*., 64, lines 10-21**.**Mr.

Brown also handled and breathed vapors from liquid condensate, a material containing between 1.0% and 9% benzene (10,000 ppm – 90,000 ppm bezene) as disclosed by a Shell memorandum listing products and materials containing more than 0.5% benzene issued in 1995 by the Shell Products, Safety and Compliance Department.

Another task performed by Mr. Brown involved use of liquid asphalt, an asphalt paint type product that he frequently used during the 1968-1975 period to coat the exterior of oil and gas piping that was laid out from the offshore platforms to the shore East Bay Central facilities. This material, sometimes referred to as "black magic," contained 1% to 3% benzene and would be applied directly by hand onto the piping. Direct skin contact was frequent and of long duration.

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found significant benzene exposure to these workers who, like the plaintiff, had exposure to benzene and also worked much of time out of doors.

These tasks involving direct contact with benzene containing materials and inhalation of vapors from those materials were at all times done without the protection of respirator masks or impermeable gloves. No warnings or precautions were communicated to Mr. Brown about benzene or benzene containing materials or products. No information about hazardous materials was provided to him either via the weekly safety meetings he attended for over twenty years or in any material safety data sheets ("MSDS"), although Shell had a respirator program and equipment for other refinery workers and had MSDS and safety training materials videos, etc. None of these precautionary measures, hazardous materials communication, or safety equipment was provided to Mr. Brown.

2. Plaintiff contests Shell's assertions that benzene cannot cause MM

(1) Shell's assertion that the majority of epidemiological studies establish no association between benzene exposure and MM is wrong. Shell Brief in Support of Motion to Exclude Plaintiff's Causation Experts, ("Shell Brief") p. 2. The meta-analysis done by Dr. Peter Infante included "all published benzene cohort studies." The results demonstrated a significant increase in MM in populations exposed to benzene versus the general population. Infante ¶¶ 18 – 19.

(2) Shell's statement, "lack of epidemiology linking crude oil and other hydrocarbons to multiple myeloma are particularly telling" is not true. Shell Brief, p.2. In fact, there are good epidemiological studies linking crude oil and other benzene containing materials cited by Dr. Infante in his expert report and in his affidavit. Infante ¶¶ 12, 13, 17 – 28. **The Delzell et al. (1992), the Divine and Hartman (2000)** and **Kirkeleit et al (2007)** studies referenced by Dr. Infante[7] all demonstrate significant association between crude oil and gas production and MM.

[7] All scientific literature referenced by the experts and cited herein are available to defendants' experts via their usual data bases, and Plaintiff will make them available to Defense counsel and the court on request.

As Delzell et al pointed out: "these associations with Oil and Gas division work are unlikely due to chance or bias." The Kirkeleit et al study demonstrated that the significant excess of MM was observed specifically in "off-shore" workers. The authors concluded: "the results suggest that benzene exposure, which most probably caused the increased risk of acute myelogenous leukemia, also resulted in an increased risk of multiple myeloma." Thus, the authors concluded that the elevated risk of MM was likely due to benzene exposure because their study demonstrated a significant increase in AML as well, the latter being a marker cancer for benzene exposure.

The **Hansen (1992)** referenced in Dr. Infante's preliminary report described an epidemiological cohort mortality study of Danish stokers exposed to coal and oil combustion products. These individuals were engaged in the stoking of coal and oiled fueled furnaces. Workers are exposed to large amounts of hot burning soot and combustion gases, including benzene, as they open the combustion chamber to stoke the coal furnaces. Their exposure from oil furnaces results from cleaning soot from the furnace. The findings demonstrate a statistically significant excess of death from MM, 4 observed deaths, SMR (significant mortality ratio) = 388 (95% CI = 106-994). The benzene exposure of oil and gas production workers is similar to coke oven exposures.

(3) The Shell motion argues at page 9 of its brief that the lack of acceptance of benzene as a cause of MM presents a powerful case, e.g., that benzene does not cause MM. Among agencies they cite the International Agency for Research on Cancer (IARC) (1982). This is misleading. IARC concluded that there was sufficient evidence that benzene was a human carcinogen. IARC concluded that benzene was toxic to the blood forming organs and that it caused leukemia in humans. IARC did not mention risk of MM in association with benzene exposure because the literature cited in the Infante report and affidavit was published subsequent

to the **IARC (1982)** review. As such, the IARC Monograph on Benzene is outdated.

(4) Shell also misstates at page 9 of its Brief the position of the United States Occupational Health and Safety Administration ("OSHA"). OSHA has authority for evaluating the toxicity of chemical exposures in the United States. In their explanation of the Final Benzene Standard, OSHA (1987) stated "Epidemiologic studies demonstrate that benzene exposure can cause leukemia, multiple myeloma and perhaps other hematopoietic and lymphatic cancers."(Page 34479 Federal Register 1987) It also stated that taking into consideration the ratio of MM to leukemia deaths (45%) in the benzene cohort studies OSHA (1987), the cancer risk to benzene exposed workers from MM could be increased by an additional 45% beyond its estimated leukemia risk. OSHA did not add the MM data to its cancer risk assessment, but concluded that "The lack of inclusion of these data gives the Agency further confidence that the projected cancer risks (based on leukemia only) would be unlikely to overestimate the risk of total cancer among workers exposed to benzene." OSHA (1987) concluded, "The new standard will substantially reduce risks from other diseases of the blood and blood-forming organs such as aplastic anemia, multiple myeloma and pancytopenia." Thus, OSHA considered benzene as a cause of MM and indicated that it could have added an additional 45% to the risk from leukemia that was associated with the final standard of 1 ppm.

(5) Shell also refers to the Environmental Protection Administration ("EPA") and the Agency for Toxic Substances Disease Registry ("ATSDRA") on pages 9 and 10 of the Shell Brief. Both reviews are significantly out dated and of suspect methodology.

(6) Shell states at page 12 of its brief the findings of the Australian Petroleum Health Watch Study 13th Report which concludes the incidence of MM is the same as in the general population. This is true, as the risk of MM lowers with each successive follow-up study. There

also is no longer any elevated risk of leukemia, which was significantly elevated in the past.[8] What they fail to mention is that three previous Australian Health Watch Reports demonstrate an elevated risk of MM, which is statistically significant for the group of workers where the exposures were highest according to the authors. The **Health Watch 9th Report by Bisby et al.** (1992), included in the Dr. Infantes report and his affidavit at ¶ 69, demonstrates a significantly elevated relative risk of death from MM among the entire cohort. There were six deaths observed versus 2.3 expected (SMR = 2.6, 95% CI = 1.0-5.7). Likewise, In the **10th Report by Bisby (1998)**, 13 cases of MM were observed among the entire cohort versus 6.8 expected: SIR = 1.9; 95% CI = 1.0-3.3 For those employed in terminal work where exposures were reported to be highest, the risk of MM was highly elevated, based on 10 cases observed and 3.1 expected, SIR = 3.2 (95% CI = 1.6-6.0). In the 11th Report for this cohort by Gun et al. (2000), an elevated incidence of MM still was observed for the entire cohort, but was more elevated among those with the highest exposures. For the entire study population 15 cases of MM were observed versus 8.91 expected, SIR = 1.68 (95% CI = 0.94-2.78). Among workers employed in terminal work, where exposures were the highest, 10 cases of MM were observed versus 3.99 expected, SIR = 2.50 (95% CI = 1.2-4.6).

(7) Shell is also wrong in claiming at page 13 of their Brief that there is no support in the scientific literature for the proposition that benzene can cause MM and that no significantly increased risk of MM had been reported in relation to benzene exposure. The publications by **Rinsky (1987)** and **Ireland et al. (1999)** demonstrate significant elevated risks of MM, and 5 out of 7 of these studies show an elevated risk.

(8) Shell cites a study at page 13 of their brief by **Wong et al. (1999)** a meta-analysis

[8] Logically, empirically associated health risks diminish as the at-risk cohorts become educated to them; e.g., the risk of lung cancer associated with smoking cigarettes.

of petroleum refinery workers as showing no elevation in MM. This analysis by Wong suffers from many methodological deficiencies; e.g., selection bias. The authors Goldstein and Shalat (2000) wrote a critique of Wong's meta-analysis for MM, stating it was like "fishing in sterile waters." They argue, since the methodology showed no increase in AML as well, it cannot be used for a categorical determination of whether benzene causes MM: the absence of AML indicates that the absence of MM cannot be reasonably relied upon to show that benzene containing materials cannot induce multiple myeloma. Typically, if AML is not elevated, MM will not be elevated. Infante ¶¶ 80 – 90.

Dr. Peter Infante's affidavit also addresses and explains why other studies Shell's brief uses to argue no association between benzene exposure and MM, Sonoda et al. (2001), Alexander et al. (2007), are unpersuasive. Infante ¶¶ 54, 56.

(9) Shell misunderstands the purpose of exploring the general causation hypothesis at issue in this case when it suggests that Plaintiff's experts rule out "idiopathic" (unascribed or unknown) causes of MM. (Shell Brief, p. 14) Because the Mr. Brown was exposed to benzene containing materials, and there is a bona fide basis for such an association, ruling out idiopathic causes is simple logic and has been done.

(10) At pages 17-18 of their brief, Shell argues that Dr. Infante cannot make an argument about the biological plausibility of benzene causing MM since he is not a toxicologist and his opinions on this are without genetic and other biological supporting evidence. Missing the point again, Shell fails to appreciate that experts can rely on the work of other specialists to support their opinions if those are reliable and relevant. Dr. Infante's biological plausibility argument is very well set out in his affidavit; it is almost the same as the conclusions reached by Goldstein in his 1990 publication. Dr. Infante also published his biological plausibility argument in his peer-reviewed meta-analysis. **Infante (2006).** Infante ¶¶ 29 – 42.

E. Plaintiff's Experts Meet the Requirements of *Daubert* and FED. R. EVID. 702.

1. <u>The testimony of Dr. Peter Infante should not be excluded</u>

Dr. Infante has held the positions of Adjunct Professor and Professional Lecturer of Environmental Health at the George Washington University School of Public Health Services. He is a Fellow of the American College of Epidemiology. In 2008, he served as expert consultant in epidemiology for the National Toxicology Programs Report on Carcinogens. In March, 2009 he served as an expert epidemiologist on a Working Group for the International Agency for Research on Cancer (IARC), which is the expert committee on causes of cancer for the World Health Organization. Prior to his appointment at George Washington University, he served for three years as an epidemiologist and health scientist for the National Institute for Occupational Safety and Health (NIOSH) where he conducted epidemiological studies of workers exposed to a number of substances, including benzene. For twenty-four years, he was employed by OSHA in Washington, D.C., and for five of those years he was Director of the Office of Carcinogen Identification and Classification. His affidavit includes a detailed description of his qualifications and a summary of the work that he has done in the past that is relevant to the questions and issues in this case; i.e., causes of multiple myeloma; the biological plausibility of benzene induced multiple myeloma; a detailed discussion of numerous epidemiological studies, including meta-analysis and studies of persons with working conditions similar to the plaintiff's work history showing the basis for evidence of benzene induced multiple myelomas, and the methodology he employed in reviewing numerous epidemiological studies. His proposed testimony is outlined in his affidavit and explains the epidemiological basis for the association of benzene exposure to MM and establishes general causation.

2. <u>The testimony of Dr. Robert Harrison should not be excluded</u>

Dr. Harrison is board-certified in internal medicine and serves on the medical faculty at the

University of California, San Francisco, He has served on numerous scientific review committees, consulted with the National Institute for Occupational Safety and Health, the United States Occupational Health and Safety Administration, served on the California Occupational Safety and Health Standards Board, and is past president of Council of State and Territorial Epidemiologists and past chairperson of the Occupational Health Section of the American Public Health Association. He has more than twenty-five years experience in clinical diagnosis, consultation and treatment of work-related injuries and diseases and estimates that he has diagnosed and treated more than 10,000 patients with work-related diseases, including many with solvent exposures and exposures to benzene. His affidavit outlines his proposed testimony supporting Dr. Infante's opinion establishing general causation.

3. <u>The testimony of Dr. Sheila Butler should not be excluded</u>

Dr. Sheila Butler is board-certified in Anatomic Pathology, Clinical Pathology, and Hemapathology, with 16 years of experience in pathology, and holds a Masters of Public Health from the Mailman School of Public Health, Columbia University. Her residency was with the New York City Department of Health and Mental Hygiene, Division of Epidemiology, Public Health Training, and her areas of focus included cancer prevention and control, quality of life measures, including pain management, for cancer survivorship and hematological neoplasm associated with occupational/toxic exposures. Her affidavit outlines her proposed testimony supporting Dr. Infante's opinion establishing general causation.

<u>Conclusion</u>

Shell's Motion to Exclude Plaintiff's Causation Experts should be denied without the need for a hearing pursuant to FED. R. EVID. 104 (a). There is simply an insufficient basis for its contention that Plaintiff's experts and their testimony would not meet the *Daubert* requirements. At the most, Shell should be required to depose videographically the general causation experts,

so that a mini-trial may be avoided and the issues concerning the general causation experts submitted on excerpts from their deposition testimony.

Dated: June 15, 2009.

Respectfully submitted,

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ATTORNEYS OF RECORDS FOR PLAINTIFF

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CERTIFICATE OF SERVICE

I hereby certify that on this the 15th day of June 2009, a true and correct copy of the above listed document was electronically filed with the Clerk of the Court and served on all parties of record using the CM/ECF System.

/s/ S. Reed Morgan
S. Reed Morgan